February 1941 school kids gas mask test drill: Windsor, England

Ministry of Defence

Medical Manual of Defence Against Chemical Agents

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SECTION I—GENERAL INTRODUCTION

CHAPTER I

GENERAL DESCRIPTION OF CHEMICAL AGENTS

1. The term "Chemical Agent" is applied to any chemical compound which, when suitably disseminated, produces incapacitating, damaging or lethal effects in man, animals, plants or materials.

2. The medical services must, however, also consider the toxic effects of various poisonous substances which may be encountered incidentally under Service conditions.

Historical

3. Chemical operations in the modern sense were first waged by Germany in the 1914–18 War. Chlorine gas was released from large cylinders in a favourable wind. The Allies were taken by surprise and, as the men had no respirators, casualties were heavy. Means of protection had to be improvised at once. The first official respirator (a cotton pad soaked in hyposulphite of soda, glycerine and sodium carbonate) was issued in May 1915, and after that date defence, on the whole, kept ahead of attack—so much so, that the use of phosgene gas by the Germans in December 1915 found the Allies relatively well protected against its effects.

In the hope of overcoming this protection the Germans tried arsenical irritant smokes which they hoped would penetrate the box respirator then used by the Allies. This proved a comparative failure. The use of mustard agent against the Allies was, however, highly successful.

4. Chemical operations were not used in World War II and various conjectures have been made as to the reason for this. It is known that the Germans had chemical agents available, and at the end of the war British and American forces discovered stocks of newer agents called "Nerve Gases". These were found to be effective in extremely low concentrations. Probably the high standard of training and preparedness of the Services and the fear of retaliation by the Allies were the main reasons why chemical agents were not used.

5. Apart from World War I, there is no record of chemical operations having been used between technically well equipped combatants, but between the wars it was known that mustard agent was used to considerable effect against the unprotected Abyssinian tribesmen and troops. Chemical operations were not used in the Spanish Civil War and, apart from the use of an unspecified chemical agent in the Yemen, they have not been used by participants in the various insurrections that have taken place since World War II.

6. The use of riot control agents ("tear gas") has, however, been extended more recently to harrass guerillas, in particular to flush them from hiding and to render places of concealment such as tunnels untenable.

7. The advent of nuclear weapons, and the fact that chemical operations were not used in World War II, do not exclude the possibility of their being used in a future war. The experiences of World War I indicate that chemical agents are useful strategic and tactical weapons. They can affect both forward and rear areas.

8. During World War I, chemical agents were used only in land weapons and not from aircraft; chemical casualties were mainly due to vapour and were largely confined to troops in the field. In a future war, chemical agents may be dispersed by other methods on to selected targets far removed from the fighting line, such as cities, dockyards and factories. It, therefore, seems probable that the nature and severity of casualties may differ from those recorded in World War I.

General Factors Influencing the Employment and Choice of Chemical Agents

9. The effective use of any chemical agent is dependent on its physical and chemical properties and on meteorological conditions.

10. For tactical purposes chemical agents may be divided into two main categories as follows:

(*a*) *Non-persistent agents* are those which remain in effective concentrations for only a short time. They are released as airborne particles of a solid, droplets of a liquid, or as true gases. They are affected by prevailing weather conditions and are quickly dispersed, so that the locality in which they have been released soon ceases to be dangerous.

(*b*) *Persistent agents* are substances which remain dangerous for some considerable time unless action is taken to destroy or neutralise them. They may be liquid or solid at normal temperatures.

11. The following meteorological factors are likely to influence the use of chemical agents:

(*a*) *Winds:* Strong winds rapidly disperse non-persistent agents in open country, although dangerous concentrations may take longer to clear from woods, dugouts and built up areas.

(*b*) *Temperature:* High temperatures increase the effectiveness of the less volatile persistent agents since high vapour concentrations are given off from them. Low temperatures may freeze persistent agents and will, in any case, increase their persistence. The danger of carrying such agents into a warm building on boots and equipment, whereupon toxic vapour will be given off, should be borne in mind.

(*c*) *Rain:* Heavy rain reduces the effectiveness of chemical agents, but does not make them impossible to use.

(*d*) *Atmospheric stability:* When the air temperature is higher than that of the ground (an inversion), agents in the vapour state will persist for longer periods than when the air temperature is lower than the ground temperature (a positive lapse rate).

Chemical Agents

12. For medical purposes, chemical agents are usually classified according to pharmacological principles, but for general service usage it is desirable to classify these agents according to their overall effects on combat effectiveness. Medical officers in field force units must understand both types of classification in order that they may be able to advise personnel of other arms. Table I shows these methods of classification on a comparative basis.

Table I. Medical and Service Classification of Chemical Agents

Medical Classification	*Equivalent Service Classification*
A. *Agents liable to be met in warfare*	
1. Nerve agents (G and V)	Lethal agents (Nerve)
2. Lung damaging agents (Phosgene and Cl_2)	Lethal agents (Choking)
3. Vesicant agents (sulphur mustard, Lewisite etc)	Damaging agents (Blister)
4. Psychotomimetic agents (LSD, BZ)	Incapacitating agents (Mental)
5. Miscellaneous agents	
(a) Cyanide, CNCl	Lethal agents (Blood)
(b) AsH_3	
6. Herbicides	Anti-plant agents
B. *Agents liable to be met in Riot Control and/or Warfare*	
7. Sensory irritant agents (CS)	Riot Control agents
8. Vomiting agents (DM)	Incapacitating agents (physical)

SECTION II—AGENTS LIABLE TO BE MET IN WARFARE

CHAPTER II

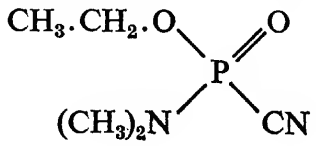
NERVE AGENTS

Introduction

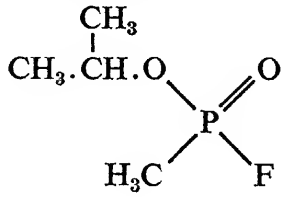
38. At the end of World War II stocks of a new type of chemical weapon were discovered in Germany. The filling, named "TABUN" by the Germans, was one of a series of compounds discovered during research on insecticides. It was found to be an acute systemic poison active in extremely low dosage, the toxic effects being similar to those caused by physostigmine and diisopropyl phosphorofluoridate (DFP). The high toxicity of the series had excited the interest of the German War Department, and work had been going on in secret since 1937.

39. Many compounds structurally related to TABUN have since been made, some of which are even more toxic. Those members of the series which are of military importance are now included in the generic term "Nerve Agents". Some of them have been given names, but they are more usually known by code letters, for example, GA, GB, GD, VX. The formulae of some of these compounds are shown below.

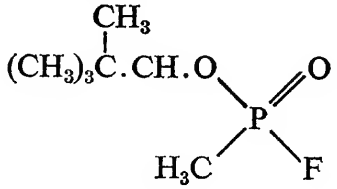
GA (Tabun)
0–Ethyl, N, N-dimethylphosphoramidocyamidate



GB (Sarin)
0–Isopropyl methylphosphonofluoridate



GD (Soman)
0–1,2, 2-Trimethylpropyl methylphosphonofluoridate



Physical and Chemical Properties

40. The nerve agents are all organo-phosphorus esters related to certain types of insecticide. They are liquids varying in volatility over a range similar to that between petrol and heavy lubricating oil. They have low freezing points, none freezing until −40°C.

(*a*) *Appearance.* Liquid nerve agents are pale yellow to colourless, but may have a slightly darker colour due to impurities. They are essentially odourless.

(*b*) *Stability.* Nerve agents are fairly soluble in water, being very slowly broken down by hydrolysis, yielding less toxic products. They are rapidly destroyed by strong alkalis and bleaching powder.

(*c*) *Powers of Penetration.* Normal clothing is penetrated by these agents whether contact is with the liquid or vapour state. Liquid agents usually penetrate by diffusion of vapour through the fabric. Leather is penetrated in the same manner as skin, but rubber, especially butyl rubber, and synthetic materials such as polythene are more resistant. Penetration of the Suit Protective NBC will not occur within 6 hours. The agent can penetrate into normally non-absorbent materials such as webbing, leather and wood and can continue to present a hazard by desorption of the vapour.

(*d*) *Persistence.* A wide variation in volatility between different members of the group leads to a wide range of persistencies. Additives may be used to alter the persistency of any one agent. The "G" agents are much less persistent than the "V" agents.

Detection

41. The agent can be detected by chemical means, and reactions which produce a colour change are made use of in equipment available for detection. The presence of liquid agent can be ascertained by using Detector Paper or Detector Powder, and of vapour by using the Residual Vapour Detector. In water, nerve agents can be detected in concentrations above 0·5 parts per million by the use of the Water Testing Kit, Poisons.

Protection

42. Ordinary clothing affords very little protection against nerve agent and special protective garments are required. The Suit Protective NBC, Gloves Protective NBC and the Respirator S6 NBC, give complete protection against these agents, in both the liquid and vapour state, for at least six hours. Before this period has elapsed, the suit and gloves should be changed. Boots which have a leather upper are slowly penetrated by the agent; additional protection can be gained by the use of overboots, and by the liberal use of fullers' earth inside the boot.

Decontamination

43. Liquid agent on the skin must be removed as soon as possible. Similarly, liquid agent must be removed from personal and unit equipment to prevent a continuing hazard.

(*a*) Decontamination of the skin is best carried out by means of the Decontamination Kit Personal No. 1. This contains pads which, when dabbed and rubbed on the skin, release fullers' earth powder. The powder soaks up the liquid and retains it by adsorption. For large areas of contamination and for items of personal equipment, such as webbing and small arms, the use of the Decontamination Kit Personal No. 2, which is a puffer bottle containing fullers' earth, is recommended.

(*b*) Expendable materials should be burned or buried. It should be remembered that if they are burnt, toxic vapours will be given off and due consideration must be given to the protection of individuals in the vicinity and downwind. Articles to be buried should be buried with a quantity of bleach slurry to ensure destruction of the agent.

(*c*) For large items of equipment such as vehicles or weapons, decontamination is best carried out by the use of the Decontaminating Apparatus NBC Portable or by scrubbing with bleach slurry. Hosing with water may remove most of the agent, but might spread contamination.

Mechanism of Action

44. Nerve agents inhibit the enzyme acetylcholinesterase. This enzyme hydrolyses acetylcholine, which is liberated when nerve impulses reach cholinergic nerve endings. The effect of a nerve agent is, therefore, to cause an individual to accumulate acetylcholine and so poison himself.

45. The parasympathetic nerve endings most obviously affected are those to the iris and ciliary body, those to the lachrymal and salivary glands, and those to the glands and muscles of the bronchial tree and gastrointestinal tract. Acetylcholine is also released in cardiac muscle from vagus stimulation and at the sympathetic nerve endings of the sweat glands. Symptoms due to an accumulation of acetylcholine at these sites are referred to as muscarinic symptoms and those due to acetylcholine accumulated at the neuromuscular junctions and the pre-ganglionic sympathetic synapses are referred to as nicotinic symptoms. In addition there are less well-defined central effects (Table II).

TABLE II

Pharmacology of Nerve Agents

Type of Action	*Site of Action*	*Response*
Muscarinic	Glands	
	Sweat, Salivary, Nasal, Bronchial, Gastro-intestinal	Increased Secretion
	Smooth Muscle	
	Bronchial	Constriction
	Cardiovascular	Bradycardia
	Iris	Miosis
	Gastro-intestinal	Increased mobility Colicky pain Diarrhoea
	Bladder	Involuntary micturition
Nicotinic	Pre-ganglionic synapses	Hypertension Pallor
	Neuromuscular junction	Weakness Muscular twitching Fasciculation Paralysis
Central	Central Nervous System	Apprehension Hyperexcitability Weakness Inco-ordination Convulsions Respiratory failure

46. The nerve agents are cumulative poisons, and repeated exposures to low concentrations, if not too widely separated, will eventually give rise to symptoms due to a gradual inhibition of acetylcholinesterase activity in the blood and tissues. Restoration of the cholinesterase activity to normal levels takes several weeks, but clinical recovery from acute effects usually takes place within a few days, due in part to a process of adaptation to lower levels of the enzyme.

Pathology

47. The damaging effects of nerve agents are on function and not on structure. Post mortem examination reveals signs consistent with death from asphyxia and there is usually evidence of blocking of the air passages with fluid secretions, if the case has not been treated with atropine, and oedema of the lungs. These, together with a decrease of acetylcholinesterase activity, which can be assayed in autopsy material, are the only objective signs.

Signs and Symptoms

48. These vary with the route and severity of poisoning. Some agents, *e.g.*, GA and GB, are normally vapours and others *e.g.*, VX, are normally liquids. The former attack principally by the respiratory route, the latter mainly through the skin.

49. *Respiratory route.* Poisoning may be mild or severe.

(*a*) *Mild poisoning.* Signs and symptoms may become noticeable within a few minutes of inhalation of even quite low concentrations of nerve agent. These are tightness of the chest, rhinorrhoea and salivation, miosis with dimming of vision and difficulty in accommodation accompanied by frontal headache.

These signs and symptoms are largely due to the local absorption of nerve agent and may be expected to persist for only a few hours, although headache and visual difficulties may last up to three days.

(*b*) *Severe poisoning.* The signs and symptoms referred to above become more pronounced; salivation and rhinorrhoea become so profuse that watery secretions run out of the sides of the mouth. Respiration becomes laboured because of obstruction from broncho-constriction and fluid in the airway, and audible wheezing will occur. Systemic effects from general absorption of the agent will become apparent leading to more marked miosis and severe sweating. Abdominal effects become prominent with profuse and uncontrollable vomiting, colicky pain and involuntary defaecation and micturition.

Muscular weakness occurs with fasciculation, convulsions and paralysis.

Death is due to asphyxia, and may occur within minutes.

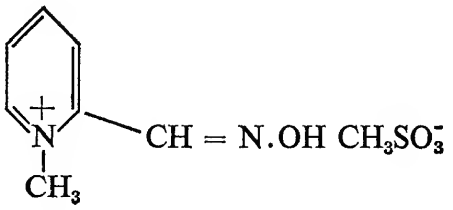
50. *Cutaneous route.* Local effects as described in para. 49a do not occur. The syndrome consists of those systemic effects described in para. 49b, preceded by general malaise. The progress of events is slower than in poisoning by the respiratory route.

51. *Gastro-intestinal route.* The onset of signs and symptoms from the ingestion of food and water contaminated with nerve agent is likely to be more rapid than the onset following skin absorption. The syndrome, however, is similar.

Treatment

52. The success or failure of treatment for nerve agent poisoning depends upon the speed with which the asphyxiating effect of the accumulated acetylcholine can be countered. This effect is due to three factors: first, paralysis of the respiratory muscles, especially the diaphragm; second, failure of the respiratory centre; and third, obstruction to air entry from broncho-constriction and the accumulated secretions. The two drugs at present used in the treatment of nerve agent poisoning are atropine and pralidoxime mesylate (oxime P_2S).

P_2S (Pralidoxime Mesylate) 2-Hydroxyiminomethyl-l-methylpyridinium methanesulphonate



To be fully effective these drugs must be used together: atropine reduces the sensitivity of the neurone or end-organ to accumulated acetylcholine; oxime, on

the other hand, acts by re-activating inhibited cholinesterase. GD, however, forms an irreversible complex with cholinesterase and is, therefore, resistant to oxime. It is important to note that the combined effects of atropine and hypoxia on the myocardium may rarely result in ventricular fibrillation.

53. The regime of treatment consists of pre-treatment, self aid, first aid and subsequent medical therapy in that order.

Pre-Treatment

54. The aim is to produce and maintain a therapeutically effective level of oxime in the blood. The dose required to achieve this is 4 g. every 6 hours. The standard pack, labelled Pralidoxime Mesylate (Oxime) Tablets, contains sufficient for 24 hours. Each dose consists of one quick release and three slow release tablets.

Pre-treatment must commence when a threat of chemical attack has been declared, and should continue for at least 36 hours after an attack with nerve agents, in order to protect against the late effects of slow absorption through the skin.

Mild loosening of the stools may be experienced by a proportion of those taking Oxime, but this does not interfere with normal activities.

Self Aid

55. (*a*) *Atropine*. The most urgent measure is the self administration of atropine immediately signs or symptoms of poisoning develop. Three automatic injection devices (Autoject), each containing 2 mg. of atropine, are carried in the respirator haversack. The second and third Autojects are used at 15 minute intervals if symptoms persist. Each injection is made through the clothing on the outer aspect of the middle of the thigh.

(*b*) *Pralidoxime mesylate* (oxime). An extra 4 g. dose of oxime should be taken following the first injection of atropine.

(*c*) *Decontamination*. Any skin exposed to liquid agent must be decontaminated.

First Aid

56. First aid must be rendered to any individual unable to aid himself: atropine injection and artificial respiration are the essential measures. The casualty's own autojects should be used.

The most effective form of artificial respiration is positive pressure: the Resuscitator NBC Portable should be used if available. Both the casualty and the resuscitator should be decontaminated as soon as possible—if necessary while artificial respiration is in progress. If no resuscitator is available, artificial respiration by mouth to mouth means can be carried out provided that the atmosphere is non-toxic and the casualty's face has been decontaminated. Manual methods (*e.g.*, Holger–Neilsen) are unlikely to be effective, but may be used if positive pressure methods cannot be employed, in which case the casualty should remain masked.

(Full details of all these methods of artificial respiration are given in Section VIII).

Artificial respiration must be continued until the casualty is breathing normally or for at least two hours.

Medical Therapy

57. Full medical treatment cannot be carried out in a toxic environment. Where possible the following regime of treatment is recommended:

(*a*) Complete decontamination of the casualty. Subject to life-saving requirements, this will consist of removal of the clothes and their disposal, and decontamination of the skin.

(*b*) A clear airway and artificial respiration must be maintained. A sucker may be required to remove excessive secretions.

(*c*) Atropine must be given until certain of the effects can be seen clinically. The required degree of atropinization is indicated by a dry mouth and a heart rate of 90–100 per minute. (Mydriasis may be an unreliable sign after nerve agent poisoning). This state should be maintained for 24 hours. The drug is conveniently administered in 2 mg. doses intravenously. Repeated doses will be needed as indicated by the pulse rate. Very large total doses, of the order of 200 mg., may be required in cases of severe poisoning. The medical officer should be alert to the signs of atropine poisoning which are a combination of central and peripheral nervous effects. The central action may produce euphoria, hallucinations, anxiety, restlessness, excitement and delirium, followed in severe cases by coma and depression of respiration. The more obvious peripheral effects are rapid pulse, dry mouth and throat, and dry hot skin. There may be hyperpyrexia. When oxime has been given after large doses of atropine, the possibility of atropine poisoning should be kept in mind.

A special preparation of atropine, containing 2 mg. in 1 ml. ampoules, is available for treatment of nerve agent or organophosphorus pesticide poisoning and this should be given by ordinary syringe and needle.

(*d*) Pralidoxime mesylate should be given concurrently in a dose of 1 g. intravenously every hour up to four injections. If the casualty's condition allows, oral dosage should be maintained. Oxime in ampoules, each containing 1 g. in 6 ml., is available for the treatment of nerve agent or organophosphorus poisoning.

(*e*) Treatment of ocular symptoms. The instillation of 1 per cent atropine eye drops or the application of 0·5 to 1 per cent atropine ointment into the eyes is more effective than parenteral atropine in relieving headache and ciliary spasm.

(*f*) If circumstances permit, treatment of severe nerve agent poisoning by curarization and intubation, after admission to hospital, should be considered.

Special Care in the Tropics

58. Since atropine inhibits sweating, extra care is required in tropical climates. Atropinization is still necessary in treating nerve agent casualties, but the possibility of heat effects must be borne in mind.

Course and Prognosis

59. The outlook depends upon the amount of agent absorbed and on the promptness and efficiency with which remedial measures are undertaken. Life can often be saved by treatment even though many times the lethal dose has been absorbed.

The function of the respiratory centre and muscular power returns, in most cases, within three or four hours. Recovery may not be immediately complete, however, and the danger of hypoxia remains for some hours due to recurrent bouts of muscular weakness.

Recovery, when it occurs, is likely to be complete in a few days, though heightened susceptibility to further exposure will persist for some weeks and tolerance does not develop.

60–61. *Reserved.*

CHAPTER III

LUNG DAMAGING AGENTS

Introduction

62. The most important member of this group is phosgene. It was used with great effect in World War I when it accounted for some 85 per cent of the deaths attributable to chemical agents.

Since the action of phosgene may be regarded as typical of that exerted by other members of this group it is the only agent discussed in this Chapter.

Other members of the group, also used in World War I are chlorine (paragraph 223) and chloropicrin (paragraph 222). Cyanogen chloride and bromide, which are classed as miscellaneous agents and discussed in Chapter VI, produce some effects similar to the lung-damaging agents.

PHOSGENE

Physical and Chemical Properties

63. Phosgene (carbonyl chloride: $COCl_2$) is a colourless gas readily condensed by pressure or low temperatures to a colourless liquid with a boiling point of 8 °C. It has an odour resembling that of new-mown hay. Phosgene reacts rapidly with water to yield non-toxic hydrolysis products.

64. Although phosgene is a non-persistent agent, the vapour is somewhat heavier than air. It may, therefore, remain in dangerous concentrations in trenches, bunkers, valleys and woods for some considerable time depending on the atmospheric conditions.

Detection

65. There is no device available for the detection of this agent.

Protection

66. Full protection is afforded by the Respirator S6 NBC.

Decontamination

67. Because of its physical properties the agent will not remain long in its liquid state. Decontamination is not, therefore, necessary.

Mechanism of Action

68. Phosgene increases the permeability of the alveolar capillaries with resultant pulmonary oedema. This interferes with pulmonary gaseous exchange leading to anoxia. The loss of fluid into the alveoli also results in haemoconcentration which, together with the anoxia, causes cardiac embarrassment which may proceed to cardiac failure.

Pathology

69. The outstanding feature of phosgene poisoning is massive pulmonary oedema. This is preceded by damage to the bronchiolar epithelium, development of patchy areas of emphysema, partial atelectasis, and oedema of the perivascular connective tissue.

The trachea and bronchi are usually normal in appearance. This contrasts with the findings in chlorine and chloropicrin poisoning in which both structures may show serious damage to the epithelial lining with desquamation.

The lungs are large, oedematous and darkly congested. Oedema fluid, usually frothy, pours from the bronchi and may be seen escaping from the mouth and nostrils.

With exposure to very high concentrations death may occur within several hours; in most fatal cases pulmonary oedema reaches a maximum in 12 hours followed by death in 24–48 hours.

If the casualty survives, resolution commences within 48 hours and, in the absence of complicating infection, there may be little or no residual damage.

Signs and Symptoms

70. On exposure to phosgene there may be some irritation of the eyes and respiratory tract. This is manifested by smarting with lachrymation, catching of the breath with coughing, choking and a sensation of tightness and pain in the chest. Nausea, retching, and vomiting may develop, interfering with the wearing of the respirator.

The severity of these initial symptoms is no guide to prognosis since casualties with severe symptoms may fail to develop any serious lung damage, whereas others with little initial irritation may later develop fatal pulmonary oedema.

Following these initial symptoms there is, usually, a latent period during which the casualty suffers little discomfort and has no abnormal chest signs. This period may last for between 30 minutes and 24 hours. Severe exertion during this time may precipitate serious or fatal respiratory or cardiac symptoms.

The latent period is followed by the development of the signs and symptoms of pulmonary oedema. These begin with rapid shallow breathing, cyanosis, and painful cough with the expectoration of increasing quantities of frothy white or yellowish liquid. As the oedema progresses, discomfort, dyspnoea and apprehension increase. Examination of the chest reveals diminished breath sounds with rales and rhonchi in all areas.

Concurrently with these symptoms haemoconcentration develops which, together with anoxia, produces cardiac embarrassment. The pulse weakens and the rate increases to the order of 130–140 beats per minute. Circulatory collapse and cardiac failure may follow.

Of the fatal cases some 80 per cent die within 48 hours of exposure. The subsequent development of bronchopneumonia accounts for a number of deaths after this period.

Treatment

71. The initial symptoms are not reliable in prognosis, and, although it may be inevitable that men who have been exposed to phosgene must continue the battle, strenuous activity predisposes to the development of pulmonary oedema.

If, however, symptoms of respiratory distress occur, indicating the onset of pulmonary oedema, the usual therapeutic measures for this condition should be commenced without delay. Initially these measures should consist of warmth, strict rest and oxygen if available.

The casualty should be kept comfortably warm, but care must be taken to avoid over-heating.

Rest may be disturbed by anxiety, restlessness and coughing. The latter may be controlled by codeine phosphate in a dosage of 30–60 mg. and the use of morphine should be considered. It is important, however, to weigh the merits of sedation against further depression of the respiratory centre.

Oxygen, the most beneficial treatment, is indicated where there is cough, dyspnoea, cyanosis or restlessness. Where possible, it should be administered initially in high concentration and at positive pressure (hyperbarically). Artificial respiration is contra-indicated.

In addition to the above, antibiotics should be given to prevent pulmonary infection.

72–73. *Reserved.*

CHAPTER IV

VESICANT AGENTS

Introduction (MUSTARD GAS)

74. These are substances which act primarily by damaging the skin, eyes, mucous membranes and the subcutaneous tissues, though remote effects may also occur after absorption into the body.

Sulphur mustard ("mustard gas") was developed by the Germans in 1917 and proved to be the most effective chemical agent used in the first World War. It is estimated that some 168,000 casualties were caused by its use. Since the first World War, sulphur mustard has been used effectively in Abyssinia, but has not been known to have been used in any other conflict.

These agents can be employed in any weapon system with resultant dissemination as liquids, aerosols or vapours.

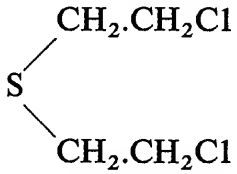
75. The vesicant agent group includes the following:

(*a*) Sulphur mustard ("mustard gas").
(*b*) The nitrogen mustards.
(*c*) The arsenical vesicants—Lewisite and "The Dicks".
(*d*) Phosgene oxime.

SULPHUR MUSTARD

Physical and Chemical Properties

76. (*a*) Sulphur mustard is Bis (2-chloroethyl) sulphide



$(ClCH_2.CH_2)_2S$

It is normally a liquid, boiling point 217°C., freezing point 14·4°C., but when encountered as a weapon of war its physical properties may be significantly altered by additives. The specific gravity of the pure substance is 1·3 and, therefore, it sinks in water leaving only a thin film on the surface.

(*b*) *Appearance.* It is an oily liquid, varying from colourless to dark brown, according to its purity.

(*c*) *Odour.* Sulphur mustard has a fairly characteristic odour reminiscent of garlic. However, detection by smell is unreliable particularly by those who are unfamiliar with it or who do not suspect its presence. Further, the sense of smell tires quickly and a rising concentration in the air may thus escape notice.

(*d*) *Solubility.* Although sulphur mustard is only very slightly soluble in water (less than 1 per cent), both the liquid and vapour are readily soluble in oils, fats and organic solvents.

(*e*) *Stability.* It is both physically and chemically stable and is unaffected by normal ranges of atmospheric temperature. It is only slowly hydrolysed by water to hydrochloric acid and thiodiglycol. Strong oxidizing agents are required to neutralize it, bleaching powder (see para. 153) being particularly effective. (N.B. If bleaching powder is used as a decontaminant it should be mixed to a slurry with water. Use of the dry powder on sulphur mustard results in spontaneous combustion).

(*f*) *Powers of Penetration.* Sulphur mustard will eventually penetrate all but the most impervious substances such as metals, glass and glazed tiles. Both liquid and vapour readily penetrate ordinary clothing, especially woollens which contain natural oils in their fibres. When droplets of

liquid agent fall on to clothing the injury that may result to the underlying skin is usually caused by vapour which has passed through the material rather than by liquid itself. Slower penetration occurs through rubber and synthetic materials such as polythene. Penetration of the Suit Protective NBC will not occur inside six hours.

(*g*) *Persistence.* The liquid agent vaporises very slowly at room temperature and is therefore very persistent. In certain weather conditions it may remain in the liquid or frozen state, giving off vapour slowly for days, weeks or even months. Additives may be used to alter its persistence. Liquid or frozen agent may be carried by boots and other items of equipment to warmer surroundings where vaporization will occur. The agent may also persist under the surface of ground which appears to be free from contamination and which may prove dangerous for years if it is disturbed. Where the agent has been absorbed by materials or structures, it will continue to present a vapour hazard by desorption.

(*h*) *Influence of Tropical Climate.* High atmospheric temperatures markedly increase the rate of vaporization of the agent; hot humid weather in particular significantly increases the rapidity and degree to which sulphur mustard affects the skin.

Detection

77. The agent can be detected by chemical means, and reactions which produce a colour change are used in the equipment available for detection. The presence of liquid can be ascertained by using Detector Paper or Detector Powder and of vapour by using the Residual Vapour Detector.

In water, sulphur mustard can be detected in concentrations of 2 parts per million and above by means of the Water Testing Kit, Poisons.

Protection

78. Ordinary clothing affords very little protection against sulphur mustard and special protective garments are required. The Suit Protective NBC and Gloves Protective NBC, when worn with the Respirator NBC S6 give complete protection against the agent, in both the liquid and vapour state, for at least 6 hours. Before this period has elapsed, the suit and gloves should be changed. Boots which have a leather upper are slowly penetrated by the agent; additional protection can be gained by the use of overboots, or by the liberal use of fullers' earth inside the boot.

Decontamination

79. Liquid agent which has come into contact with the skin must be removed as soon as possible. Similarly, liquid agent must be removed from personal and unit equipment to prevent a continuing hazard.

(*a*) Decontamination of the skin is best carried out by means of the Decontamination Kit Personal No. 1. This contains pads, which, when dabbed and rubbed on the skin, release fullers' earth powder. The powder soaks up the liquid and retains it by adsorption. For large areas of contamination and for items of personal equipment, such as webbing and small arms, the use of the Decontamination Kit Personal No. 2, which is a puffer bottle containing fullers' earth, is recommended.

(*b*) Expendable materials are best dealt with by burning or burying. It should be remembered that if they are burnt toxic vapours will be given off and due consideration must be given to the protection of individuals in the vicinity and the downwind hazard. When articles are buried they should be buried with a quantity of bleach slurry to ensure destruction of the agent. OR, SCRUB EQUIPMENT WITH BLEACH SLURRY

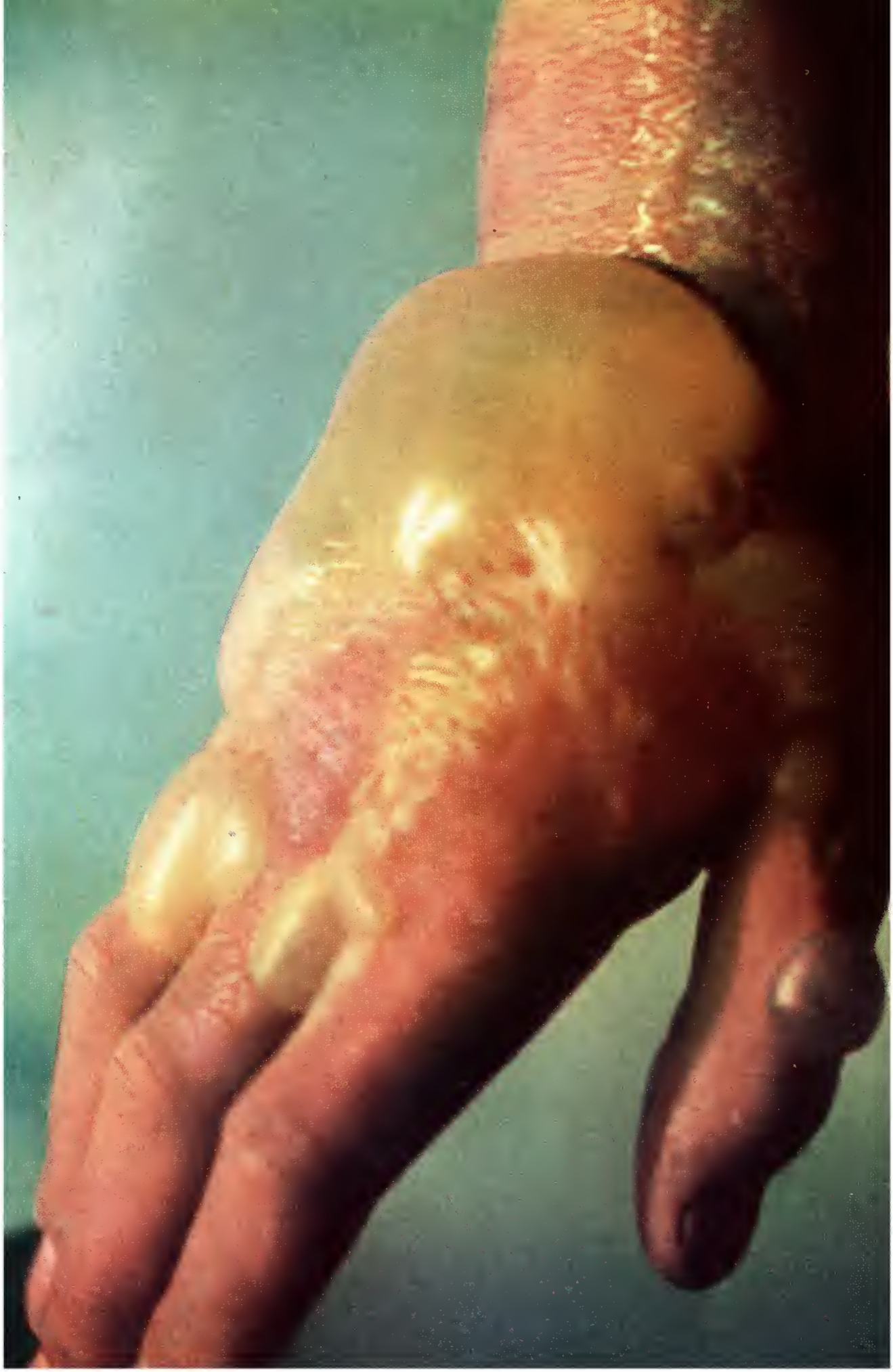


Plate III. Effect of Liquid Sulphur Mustard on the Skin.
Blisters appearing on the skin 24 hours after exposure to liquid sulphur mustard.

CHAPTER V

INCAPACITATING AGENTS

Introduction

99. Incapacitating agents are substances which impair the subject's ability to carry out his duties, but the use of which does not incur serious risk of death or permanent injury. Lethal agents in sub-lethal doses and blister agents, both of which may cause permanent injury, and riot control agents are excluded from this category.

100. Incapacitating agents are classified as physical incapacitants or psychotomimetic agents according to whether their action is predominantly upon the physical or mental activities of the subject.

PHYSICAL INCAPACITANTS

101. Possible mechanisms of physical incapacitation are many, but the criterion that no serious risk of death or permanent injury should result means that no practical physical incapacitant is known at present, although the vomiting agent D.M. (See Chapter VII) is described as a physical incapacitant in the Service Classification (Table I).

PSYCHOTOMIMETIC AGENTS

102. There are many drugs which act upon the central nervous system to produce incapacitation; few of these are sufficiently potent and safe, or possess the necessary chemical and physical properties, to make them potential chemical agents. Of these few, BZ, an atropine-like drug, is the most important, but lysergic acid diethylamide (LSD 25) and other similar drugs, merit consideration.

BZ

Physical and Chemical Properties

103. BZ is a crystalline solid at normal temperatures and sufficiently stable to be disseminated as a smoke from a pyrotechnic device.

Detection

104. There is no device at present available for the detection of this agent.

Protection

105. Full protection is afforded by the Respirator S6 NBC and Suit Protective NBC.

Mechanism of Action

106. BZ acts by blocking the activity of cholinergic synapses in a manner similar to that of atropine. Unlike atropine, BZ produces predominantly central rather than peripheral effects.

Signs and Symptoms

107. In 1–2 hours after exposure, BZ produces atropine-like effects, such as dilation of the pupils, dry mouth and increased heart rate, followed later by ataxia and drowsiness. These effects, apart from the mydriasis, give way after 6 or 7 hours to a confused mental state, in which delusions, hallucinations and aimless behaviour are common, and may persist for several days. During this phase the subject may injure himself and others. Memory for the period of the intoxication may be lost or fragmentary. The mydriasis may persist for 3 days.

Treatment

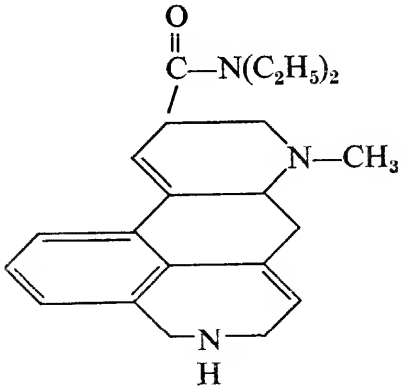
108. In the majority of casualties, symptomatic treatment is all that will be necessary. Firm restraint when necessary and a friendly attitude are called for, especially in dealing with those subjects who are capable of walking. All dangerous objects must be removed and anything likely to be swallowed should also be kept from the subject as bizarre delusions may occur. Body temperature should be observed, as heat stroke may occur, especially in tropical climates. Fluid intake must be maintained.

Physostigmine, which is used as an antidote to BZ, should be reserved for casualties who appear to be in danger. Where this treatment is deemed to be necessary, an injection of 2–3 mg. will be required to alleviate the condition. Repeated injections at intervals of 15–30 minutes may be required to avoid relapse.

Neostigmine, because it does not penetrate the central nervous system as well as physostigmine, is considerably less effective.

LSD 25

LSD 25 D-Lysergic acid diethylamide



Signs and Symptoms

109. The clinical manifestations of LSD intoxication often include an early stage of nausea, followed 45–60 minutes after dosage by a confused state in which delusions and hallucinations are common, but not always experienced. There is some evidence that the effects may be held off, at least for a time, by determination to continue duty, and that the presence of undrugged comrades enables affected subjects to maintain contact with reality. Recovery is spontaneous and is usually complete within 12 hours.

Treatment

110. The best treatment known at present for LSD intoxication is the administration of sodium amytal (200–400 mg. intravenously) to sedate the patient until spontaneous recovery occurs. Chlorpromazine has also been suggested for therapy, but does not appear to have any advantage over sodium amytal.

OTHER DRUGS

111. The phenothiazines and cannabinols, although they seem to act primarily by depressing the central nervous system, are not considered likely to be used in warfare owing to the relatively large doses required to produce an effect.

112–113. *Reserved.*

CHAPTER VI

MISCELLANEOUS AGENTS

Introduction

114. There are a number of agents which do not readily lend themselves to pharmacological classification; these include cyanide type agents and arsine. This Chapter considers the former type; arsine is discussed in Chapter XIII.

The use of cyanide agents was initiated by the French in 1916 with the employment of shells filled with hydrogen cyanide. However, because of its extreme volatility and the fact that the vapour is lighter than air, it was found almost impossible to establish a lethal concentration in the field by this means of delivery. In an attempt to overcome this disadvantage the related substances, cyanogen chloride and cyanogen bromide, were produced, the vapours of which are several times heavier than air.

With modern weapon systems, it is certainly possible to produce a lethal field concentration of hydrogen cyanide and therefore knowledge of the effects of this agent is essential.

115. The agents in this group, known also as "Blood Agents", are:

(*a*) Hydrogen cyanide.
(*b*) Cyanogen chloride (CNCl)
(*c*) Cyanogen bromide.

The latter two compounds, after absorption from the lung, react with haemoglobin in such a way that hydrogen cyanide is eventually released. Their effects on the body, therefore, are essentially similar to those of hydrogen cyanide. The rest of this chapter deals with hydrogen cyanide, but the main points of difference of the other two agents are indicated.

HYDROGEN CYANIDE

Physical and Chemical Properties

116. Hydrogen cyanide is a clear, colourless liquid with a boiling point of 26°C. It is very volatile and the vapour, being somewhat lighter than air, disperses rapidly after release. It has a smell of bitter almonds which may be noticed in concentrations as low as 1 part per million. This is well below the danger level, but is unreliable as a means of detection. Hydrogen cyanide is soluble in water producing a weak acid solution.

Detection

117. At present there is no automatic device available to the Services for the detection of these agents in the vapour state, but a Draeger tube can be used. Cyanide in water can be detected in a concentration of 20 parts per million using the Water Testing Kit, Poisons.

Protection

118. Full protection against these agents is afforded by the Respirator S6 NBC and the Suit Protective NBC. However, these agents seriously impair the effectiveness of the respirator filter which is best changed after a single exposure to them.

The protective suit is necessary for full protection since the agents, in their liquid state, can be absorbed through the skin.

Decontamination

119. Because of its physical properties the agent will not remain for long in its liquid state. Decontamination should not, therefore, be necessary.

Mechanism of Action

120. The cyanide ion reversibly complexes with the respiratory cytochrome oxidase enzyme system which results in impairment of cellular oxygen utilisation. The central nervous system, particularly the respiratory centre, is especially susceptible to this effect and respiratory failure is the usual cause of death.

Pathology

121. With exposure to high concentrations sufficient hydrogen cyanide may be inhaled in a few breaths to cause immediate death by respiratory failure. In these cases no pathological changes are demonstrable. The blood remains well oxygenated and the skin has a pink colour similar to that seen in carbon monoxide poisoning.

In cases where death is delayed, following exposure to lower concentrations, small areas of haemorrhage and softening of the brain may be seen due to anoxic damage.

Where exposure is to sub-lethal concentrations, cyanide is detoxicated in the body to harmless thiocyanate. This reaction is catalysed by intracellular transsulphurase enzymes, one of which requires thiosulphate as a substrate. The limiting factor in cyanide detoxication is the amount of available intracellular reducing sulphur which can serve as, or be transferred into, substrate. This reducing sulphur is available, *in vivo*, in the form of thiosulphate, cystine and cysteine.

In addition to the systemic effects outlined above, cyanogen chloride and bromide also have local irritant effects on the eyes and the respiratory tract similar to those of the choking agents. There may be severe inflammatory changes in the bronchioles with congestion and pulmonary oedema.

Signs and Symptoms

122. The more rapidly the tissue cyanide levels build up, the more acute are the signs and symptoms of poisoning and the smaller is the total absorbed dose required to produce a given effect.

In high concentrations there is an increase in the depth of respiration within a few seconds. This stimulation of respiration may be so powerful that a casualty cannot voluntarily hold his breath. Violent convulsions occur after 20 to 30 seconds with cessation of respiration within 1 minute. Cardiac failure follows within a few minutes.

With lower concentrations the early symptoms are weakness of legs, vertigo, nausea and headache. These may be followed by convulsions, and coma which may last for hours or days depending on the duration of exposure to the agent. If coma is prolonged, recovery may disclose residual damage to the central nervous system manifested by irrationality, altered reflexes and unsteady gait, which may last for several weeks or longer; temporary or permanent nerve deafness has also been described.

In mild cases there may be headache, vertigo and nausea for several hours before complete recovery.

With cyanogen chloride and bromide the above systemic effects are modified by their irritant properties. Exposure is followed by intense irritation of the eyes, nose and throat, with tightness of the chest and coughing. Severe lachrymation and blepharospasm may occur. Vertigo, headache and dyspnoea follow which may proceed to convulsions, coma and death. In non-fatal cases pulmonary oedema often develops, which gives rise to a persistent cough with frothy sputum, severe dyspnoea, and marked cyanosis.

Treatment

123. The success or failure of treatment for acute cyanide poisoning depends upon the speed with which cellular oxygen utilisation can be restored. This may be facilitated either by the production of methaemoglobin which also complexes with the cyanide ion, or by the introduction of thiosulphate which assists the detoxication to harmless thiocyanate. Recent work has indicated, however, that

immediate treatment with dicobalt edetate, which fixes the cyanide ion directly, is more effective.

Where possible, treatment should be initiated by the intravenous injection of 20 ml. (300 mg.) of a solution of dicobalt edetate ("Kelocyanor"). Several ampoules may have to be given if the patient does not rapidly recover consciousness. Failing this, an intravenous injection of 10 ml. of a 3 per cent solution of sodium nitrite should be given, followed through the same needle by 25 to 50 ml. of a 50 per cent solution of sodium thiosulphate.

Where intravenous therapy is not immediately available, the first step in treatment is the inhalation of amyl nitrite and two ampoules should be crushed in the hollow of the hand and held close to the casualty's nose. Artificial respiration should be commenced, if respiration has ceased or is feeble, to maintain ventilation and thus facilitate the inhalation. The dose of amyl nitrite should be repeated every few minutes to a total of eight ampoules.

Oxygen, if available, should be administered preferably by positive pressure, but because of the danger of explosion when oxygen is mixed with amyl nitrite, the latter should not be given under an oxygen face mask.

In most cases of exposure to hydrogen cyanide there is either rapid death or prompt recovery. With cyanogen chloride and bromide the same applies as far as the systemic effects are concerned. Pulmonary effects may develop immediately or be delayed until after the systemic effects have subsided. These effects should be treated in the same way as phosgene poisoning. (See Chapter III).

124–125. *Reserved.*

FURTHER READING :—

Cobalt Compounds as Antidotes for Hydrocyanic Acid.
Lovatt Evans, C. (1964) Br. J. Pharmacol. and Chemotherapy **23,** 455.

Successful Treatment of Cyanide Poisoning.
Bain, J. T. B, and Knowles, E. L. (1967) Br. Med. J. **2,** 763.

Medical Cover Required in Large Scale Production of Cyanide and Hydrocyanic Acid.
Knowles, E. L. and J. T. B. Bain (1968) Chemistry and Industry 24 Feb. 1968, 232.

Treatment of Chemical Agent Casualties USA.
Dept. of Army, Navy and Air Force.
TM 8–285 NAUMEDP–5041 AFM 160–12/15 Jan. 1968.

SECTION III—AGENTS LIABLE TO BE MET IN RIOT CONTROL AND/OR WARFARE

CHAPTER VII

RIOT CONTROL AGENTS

Introduction

126. Riot control agents are chemicals that produce irritating or disabling effects when in contact with the eyes, or when inhaled. Although there is an overlap in the symptoms produced, they may be classified as sensory irritant agents and vomiting agents.

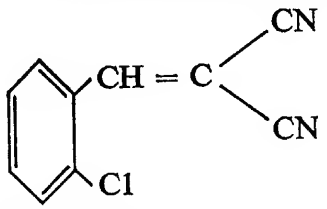
Although their primary use is for riot control, some of them may be encountered in the course of military operations. Sensory irritant agents are also used in training.

SENSORY IRRITANT AGENTS

General

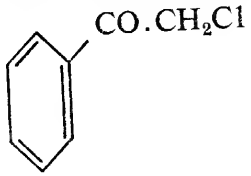
127. The most important member of this group is *o*-chlorobenzylidene malononitrile, commonly known as CS.

CS *O*-chlorobenzylidenemalononitrile



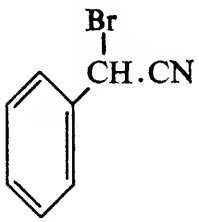
Other substances used as tear agents are (ω-chloracetophenone (CAP or CN) and bromobenzyl cyanide (BBC) which differ somewhat from CS in their physical and chemical properties; these agents are not used by British troops.

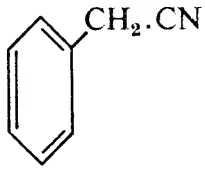
CN ω-chloracetophenone



BBC Mixture of 70% phenylbromoacetonitrile and 30% phenylacetonitrile

Phenylbromoacetonitrile Phenylacetonitrile





CS acts more quickly, is more potent and less toxic than the other riot control agents. No death or serious injury due to CS has been authenticated, whereas high concentrations of CN have caused corneal scarring, and a small number of deaths have occurred from its use in confined spaces. Further discussion in this chapter is concerned primarily with CS, but details of treatment will apply equally to the effects of CN.

128. *Reserved.*

SECTION IV—PUBLIC HEALTH ASPECTS

CHAPTER VIII

CONTAMINATION OF FOOD AND WATER

Introduction

141. Food and water cannot easily be decontaminated. Supplies must, therefore, be protected as completely as possible.

Food and water may be contaminated by chemical agents whether in the vapour, aerosol or liquid state. The most dangerous contamination is from nerve and blister agents since these are likely to be disseminated as liquids and they are more readily absorbed by foodstuffs. Exposure to high concentrations of the vapours of other agents may make food unpalatable or unfit for consumption.

FOOD

Susceptibility to Contamination

142. Nerve and blister agents are readily soluble in oils and fats. Food with a high fat content can absorb large quantities of these agents when exposed to liquid or vapour, which may diffuse throughout the material.

Liquid agents will also penetrate foods of low fat content, rendering them dangerous, but foods of this type may not absorb significant quantities of vapour.

Foods of high water content, contaminated by agents which are easily hydrolysed, may be made unpalatable by the formation of acid products of hydrolysis.

Protection of Food

143. Liquid or vapour may penetrate wooden and cardboard boxes, or paper wrappings, in sufficient quantities to make consumption of food within these packages dangerous. Sealed polythene will give good protection against vapour, but is penetrated by liquids in minutes to hours depending on the thickness of the material. Only food sealed in impervious containers, such as tins, glass or glazed earthenware jars, and foil wrappings, is completely protected against chemical agents.

Decontamination

144. When it is known or suspected that impervious containers have been contaminated they must be thoroughly decontaminated before being opened. (See Chapter IX).

If it is known or suspected that other types of container have been contaminated, the contents must be assumed to be contaminated. Where contamination is with liquid nerve or blister agent the whole contents must be condemned. Certain food contaminated by chemical agent *vapours* can be rendered safe by exposure to the air followed by cooking. (See Table III). *If there is any doubt that a particular food is safe to eat it must be condemned.*

WATER

145. Open water sources may become contaminated by direct chemical attack on an area, or by the catchment of water from such an area. In either case, concentrations sufficient to produce casualties may result. Water from deep sources, such as springs or wells, is less likely to be contaminated.

TABLE III

Effects of Chemical Agents on Food

		High Fat Content (Butter, fats, milk, cheese, meat, bacon, etc. and shell eggs).	Low Fat High Moisture Content (Fruit, vegetables, sugar, salt, etc.).	Low Fat Low Moisture Content (Cereals, tea, coffee, flour, bread, rice, etc.).
Nerve Agents	Liquid	All foods to be condemned.		
	Vapour	To be condemned.	Dry foods should be exposed to the air for 48 hours. Other foods should be washed with 2 per cent sodium bicarbonate solution, peeled where applicable, and cooked by boiling.	
Blister Agents	Liquid	All foods to be condemned.		
	Vapour	To be condemned.	As for foods contaminated with nerve agent vapour.	
Choking Agents		Agents decompose rapidly on contact with water. Food should be washed with water where possible and exposed to the air for 24 hours. Food may be made unpalatable by acid products of hydrolysis.		
Cyanide—Type Agents		Unlikely to produce dangerous contamination of foodstuffs.		
Riot Control Agents		Food may be made unpalatable to the extent of being inedible.		

SECTION V—DECONTAMINATION

CHAPTER IX

DECONTAMINATION

Introduction

149. Decontamination is a difficult and lengthy process, the need for which should be minimised by keeping personnel and equipment under cover whenever possible. This will reduce the chance of contamination by direct attack or by pick-up from contaminated objects.

150. Chemical agents of low volatility, which have been disseminated in the liquid state, can continue to present a hazard and cause casualties for days, weeks or even months. To minimise this hazard, decontamination of personnel and equipment must be carried out as soon as possible.

DECONTAMINATION OF PERSONNEL AND EQUIPMENT

Stages of Decontamination

151. There are three stages:

(*a*) *Immediate Decontamination.* This is the removal of chemical agent from exposed parts of the body after a liquid attack, and from those items of personal equipment which come into contact with the body. It follows the immediate action drill (see Appendix A) as soon as the operational situation allows. *To be fully effective, decontamination of the skin must be completed within five minutes of contamination.* The drill for carrying out immediate decontamination is fully described in Appendix A to this chapter.

(*b*) *Operational Decontamination.* The aim of this stage is to reduce the hazard from gross contamination of protective clothing caused by contact with contaminated equipment. It is a continuing process whenever time and the operational situation allow. It consists particularly of decontamination of those parts with which contact is probable during use, operation or maintenance of vehicles or equipment.

(*c*) *Unit Decontamination.* Here the aim is to remove the contamination completely from all the unit's equipment. This is a major task which cannot be performed whilst maintaining an operational role.

Methods of Decontamination

152. Decontamination can be effected by:

(*a*) Destroying the agent by chemical or physical means.

(*b*) Removing the agent by using solvents or adsorbents, or by washing.

(*c*) Weathering.

In addition the agent can be rendered harmless by sealing contaminated articles in impermeable containers or by burying them deep in the ground.

153. Destruction

(*a*) *Chemical*

(1) Bleaching Powder is issued as a general purpose decontaminant. It destroys all known chemical agents. For use it should be mixed to a slurry with water and applied by brushing on to the surface to be decontaminated. Bleaching Powder must not be used dry, since if it comes into contact with certain agents it catches fire.

TABLE IV

Summary of Agents, Properties, Methods of Recognition and First Aid

Agent	Recognition	Clinical Effects	Self Aid	First Aid	Remarks
Nerve Agent G agent—(non-persistent) V agent—(persistent)	Colourless gas and colourless to pale yellow liquid. Detector paper or powder change colour in presence of liquid. For vapour, use Residual Vapour Detector and in water use Water Testing Kit, Poisons.	Tightness of chest; headache; rhinorrhoea and salivation; miosis and dimming of vision; nausea and vomiting; sweating; convulsions; dyspnoea; respiratory failure.	Atropine by autoject immediately evidence of poisoning. Repeat twice at 15 minute intervals if symptoms persist. Extra 4 g. dose of oxime taken following injection of atropine. Decontamination of skin exposed to liquid agent.	Atropine by autoject and artificial respiration if necessary. Inject oxime. Decontamination of casualty and resuscitator.	Spced is vital in treating casualties. Atropine must be given as soon as possible. Personnel at risk should already be taking 4 g. oxime every 6 hours.
Lung damaging Agent Phosgene (non-persistent)	Colourless gas which may form white cloud. Smell of new-mown hay. No device available for detection.	Lachrymation; coughing; choking; tightness in the chest with pain. Nausea and vomiting. Latent period 30 minutes —24 hours followed by signs and symptoms of pulmonary oedema. Haemoconcentration, anoxia, circulatory collapse.		Warmth, strict rest and oxygen if available. Coughing controlled by linctus codeine.	Initial symptoms not of reliable prognostic significance.
"Cyanide-type" Agents Hydrogen cyanide (non-persistent)	Colourless gas or volatile liquid. Smell of bitter almonds. No device available to detect vapour. Detected by Water Testing Kit, Poisons.	Mild cases: headache, nausea and vertigo. Higher concentrations, in addition, convulsions and coma. High concentrations: increase in depth of respiration; violent convulsions and cessation of respiration within 1 minute.		Artificial respiration preferably by positive pressure and oxygen if available. Intravenous injection of 20 ml. dicobalt edetate or 10 ml. of 3 per cent sodium nitrite followed by 25–50 ml. of 50 per cent sodium thiosulphate. Amyl nitrite inhalations.	Usually there is either rapid death or prompt recovery. Speed in treatment is most urgent. Canister life of respirator shorter than for other agents.
Cyanogen chloride Cyanogen bromide (non-persistent)		Above systemic effects modified by irritant properties involving eyes, nose and throat with tightness of chest and coughing.		As above.	

TABLE V

Summary of Clinical Effects of Chemical Agents and their Differential Diagnosis

1. Skin

(*a*) Colour

- Grey or cyanosed
  - *Lung damaging agent*—(late effects) respiration increased, cough, pain and dyspnoes, followed after latent period by expectoration and evidence of pulmonary oedema (transient cough—*sensory irritants* and *vomiting agents*).
  - "*Cyanide type*" *agent*—respiration slowed with increased depth, followed by cessation of respiration.
  - *Nerve agent* (late effects)—Laboured respiration from broncho-constriction and fluid in airway.
- Erythema
  - *Sensory irritant agent*—transient blotchy pattern, especially on moist areas on exposure to high concentrations (transient effect on eyes and respiratory tract).
  - *Vesicant agent*—prolonged effect followed by vesication and/or blistering. (May be associated with severe effects on the eyes and respiratory tract).

(*b*) Sweating

- Excessive—*Nerve agent*—also salivation, rhinorrhoea and excessive bronchial secretions (transient salivation and rhinorrhoea from *sensory irritants* and *vomiting agents*).
- Diminished—*Psychotomimetic agent* (BZ).

2. Eyes

(*a*) Red and watering

- *Sensory irritant agents and vomiting agents*—transient effects only.
- *Vesicant agents*—also oedema of eyelids, severe conjunctivitis and oedema of cornea. Temporary or permanent blindness.

(*b*) Pupil

- Miosis—*Nerve agent*.
- Mydriasis—*Psychotomimetic agent* (BZ).

3. CNS Effects

- (*a*) Mental state
  - —Disinterested, aloof and unresponsive to commands—*Psychotomimetic agent e.g., LSD.*
  - —Confused with delusions and aimless behaviour
    - —*Psychotomimetic agent e.g., BZ.*
    - —"*Cyanide-type*" *agent* (delayed effect with residual brain damage).
  - —Depression—*Vomiting agent*, severe effect.
- (*b*) Neuromuscular
  - —Unsteady gait and weakness
    - —*Vomiting agent*, associated with sensory irritant effects.
    - —*Psychotomimetic agent LSD or BZ*, associated with mental symptoms.
    - —"*Cyanide-type*" *agent*, with residual brain damage.
  - —Muscular weakness with fasciculation—*Nerve agent*, associated with convulsions.
  - —Paralysis—*Nerve agent.*
- (*c*) Convulsions
  - —Terminal
    - —*Nerve agent.*
    - —"*Cyanide-type*" *agent*, severe effects.
  - —Delayed—"*Cyanide-type*" *agent*, mild effects.

4. Abdominal Effects

- (*a*) Vomiting
  - —*Nerve agent*—profuse and uncontrollable, with other effects of systemic poisoning.
  - —*Vomiting agent*—associated with nausea and vertigo, and sensory irritant effects.
  - —*Lung damaging agent*—associated with excessive coughing.
  - —*Sensory irritant agent*—in high concentrations associated with excessive coughing.
  - —"*Cyanide-type*" *agent*—occasionally seen in mild cases associated with nausea and vertigo.
- (*b*) Incontinence of urine or faeces—*Nerve agent* in near lethal exposure.